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IMAGE | COLON

Development of Mucormycosis Colitis during Prolonged Hospitalization

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CASE REPORT

A 50-year-old woman with previously well-controlled type-2 diabetes developed hematochezia during hospitalization for cerebral vasculitis. She had been treated with intravenous methylprednisolone (1,000 mg) for three days, requiring an insulin drip before transitioning to daily maintenance prednisone (60 mg). She was hemodynamically stable with a soft, tender abdomen on exam. An abdominal/pelvic computed tomography (CT) scan demonstrated mucosal and colonic wall thickening with edema around the transverse, descending, and rectosigmoid colon. Colonoscopy revealed multiple non-bleeding ulcers 10–18 mm in size in the rectum, sigmoid, and descending colon, with normal surrounding tissue (Figure 1); the procedure was aborted given the extent of ulceration. Pathology from biopsies taken during the procedure confirmed mucormycosis (Figure 2). Amphotericin B and caspofungin were initiated, while prednisone was tapered off to minimize further immunosuppression. The patent experienced recurrent hematochezia with progressively worse pancolitis on repeat CT of the abdomen and pelvis. Given her increasingly worse prognosis despite medical management and the unlikelihood of surviving an aggressive colectomy, colorectal surgeons recommended against surgical intervention. She ultimately chose to transition to hospice care.

This case of colonic mucormycosis presented two weeks into hospitalization compared to previously documented cases with symptoms secondary to mucormycosis presenting on admission or after an in-hospital abdominal procedure.[1-3] Gastrointestinal mucormycosis is uncommon, representing 4–7% of systemic mucormycosis cases, with a mortality of 85%.[4,5] Mucormycosis develops in the gastrointestinal (GI) tract iatrogenically or by ingesting fermented foods and herbal/homeopathic remedies.[4] The most common site of GI infection is the stomach, followed by colon, ileum, duodenum, and jejunum.[5] Presentation is variable with nonspecific symptoms such as abdominal pain, nausea, fever, and hematochezia; more severe infections present with bowel perforation.[5] Neutropenia, corticosteroids, history of organ or stem cell transplantation, acquired immunodeficiency

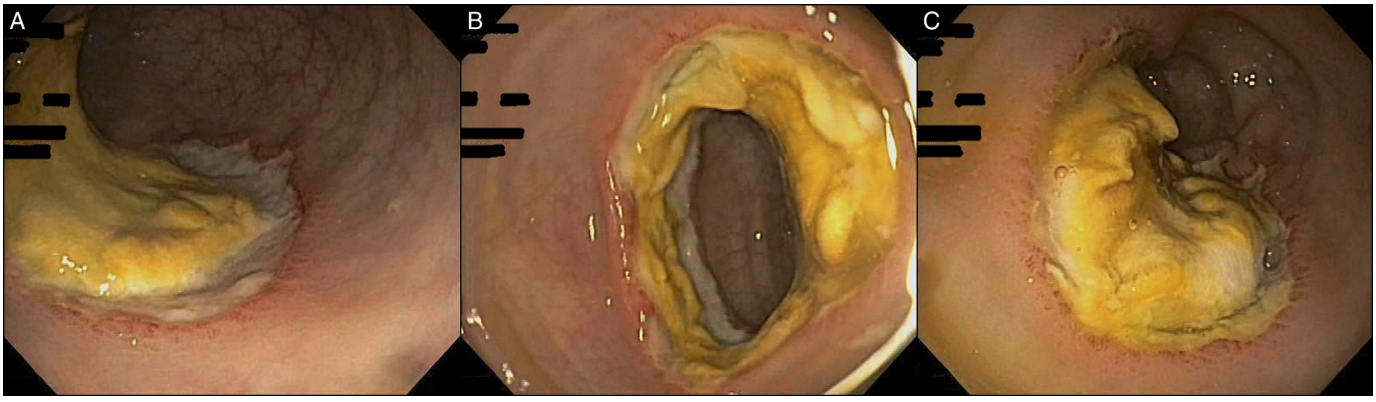

Figure 1. Colonoscopy showing the (A) descending colon lesion, (B) circumferential descending colon lesion, and (C) sigmoid lesion.

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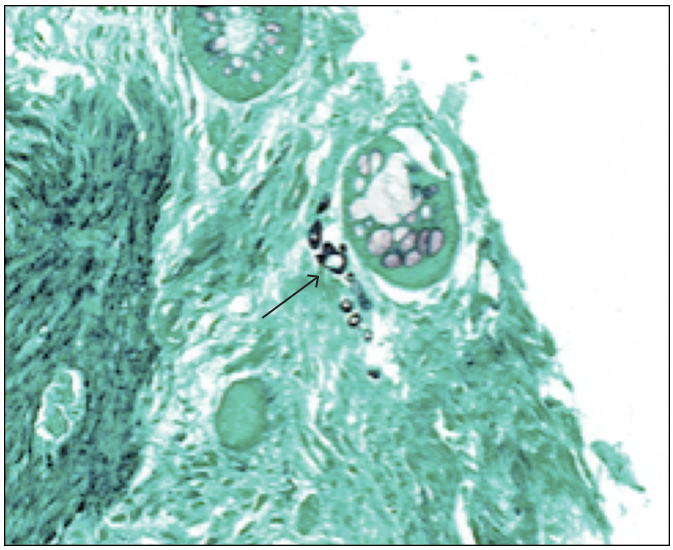

Figure 2. Grocott's methenamine silver stain of tissue-invasive fungal forms.

syndrome, and uncontrolled diabetes mellitus are among immunocompromising risk factors for infection. Diabetes is reported as the predisposing factor in 36% of cases, as uncontrolled hyperglycemia promotes further fungal growth.[4] The disease progresses by fungal invasion of blood vessels, leading to thrombosis and necrosis.[4,5] Given its invasive nature, mortality is commonly due to bowel perforation, peritonitis, sepsis, and GI hemorrhage. Reversing predisposing factors alongside medical and surgical management are the mainstays of treatment.[5] Amphotericin B is first-line treatment, although posaconazole, rifampicin (with amphotericin B), and colisitin have been utilized.[5] While early treatment provides the best outcomes, nonspecific presentations without pathognomonic endoscopic findings often delays diagnosis and treatment.[4] Gastrointestinal mucormycosis is a life-threatening disease that is important for clinicians to consider when managing susceptible patient populations.

DISCLOSURES

Author contributions: AJ Kruger wrote the manuscript and is the article guarantor. RM Modi and EE Ugbarugba wrote the manuscript and approved the final version. M. Yearsley provided the pathological images.

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